Within-Subject Association Between Daily Energy Availability and Next-Night Nocturnal Heart Rate Variability in Active Adults

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Abstract

Introduction: Low Energy Availability (Low EA, LEA, <30 kcal·kg⁻¹ fat free mass·day⁻¹) is the underlying mechanism of Relative Energy Deficiency in Sport (REDs), yet its acute autonomic correlates at the day-to-day level remain insufficiently characterized.

We quantified the within-person association between daily EA and next-night nocturnal Heart Rate Variability (HRV) in 22 physically active individuals (BUT CGM dataset) who experienced both LEA and adequate EA (≥45 kcal·kg⁻¹ fat free mass·day⁻¹) days during 8–10 days of free-living monitoring (123 person-day observations; 71 LEA, 52 adequate EA).

Methods: Daily EA was computed as (energy intake-exercise energy expenditure)/fat-free mass. Exercise energy expenditure was estimated from a cardiopulmonary exercise testing (CPET) calibrated zone-based model using individual ventilatory thresholds. Nocturnal HRV was extracted from single-lead ECG during sleep, computed on 5-minute segments and averaged across the night; each night was paired with the preceding day's EA.

Results: Using linear mixed-effects models with random intercepts for participants, eight of nine HRV parameters were significantly elevated on adequate EA versus LEA days: pNN50, HF power, RMSSD, standard deviation of heart rate (SDHR), SDNN, LF power, lnRMSSD, and RR intervals. The LF/HF ratio did not differ.

Conclusion: A single day of LEA was followed by reduced nocturnal cardiac autonomic activity compared with the same individual's Adequate EA days. These findings suggest that adequate daily energy availability may be important for next-night parasympathetic recovery. Although nocturnal HRV cannot replace direct assessment of EA, repeated overnight measurements may provide a useful non-invasive signal of recent energy status.

1. Introduction

Energy availability (EA) is the amount of dietary energy remaining for basic physiological functions after accounting for exercise energy expenditure, typically expressed relative to fat-free mass (kcal·kg^{-1} fat free mass·day^{-1}) [1]. Sustained reductions of EA below approximately 30 kcal/kg^{ffM}/day define Low EA (LEA), the underlying mechanism of Relative Energy Deficiency in Sport (REDs) [2]. Although REDs is a recognized health risk in athletic populations, the acute day-to-day effects of EA fluctuation on cardiac autonomic regulation remain poorly characterized.

Heart rate variability (HRV) is a widely used non-invasive marker of cardiac autonomic modulation [3,4]. Nocturnal HRV, recorded during sleep, removes confounding from posture, physical activity and psychological stress, providing a comparatively clean window on tonic autonomic balance [5,6]. Attenuated HRV has been reported during periods of functional overreaching and non-functional overtraining [7], and chronic energy deficiency in athletes is accompanied by a blunted sympathoadrenal response to exercise [14]. However, most prior studies used between-subject comparisons in small cohorts, susceptible to confounding by differences in baseline fitness, body composition, age and sex.

A within-subject design, comparing the same individual across different EA days, eliminates these constitutional confounders and isolates the acute within-person component of the EA–HRV relationship. Using the BUT CGM dataset [9], we quantify the within-person HRV response to the Low-vs-Adequate EA contrast in

participants who experienced both during 8–10 days of monitoring, in absolute units and as standardized effect sizes.

2. Methods

2.1. Participants

Data originated from the BUT CGM dataset [9], which combines among other single-lead ECG (1,000 Hz), smartphone food diaries, sport-watch training logs with manually determinated intensity zones, participant-reported sleep windows and laboratory cardiopulmonary exercise testing (CPET) with ventilatory thresholds annotation, collected under free-living conditions over 8–10 consecutive days. The analysis included 22 physically active participants (16 men, 6 women) who experienced both LEA days (<30 kcal/kg^{ffM}/day) and Adequate EA days (≥45 kcal/kg^{ffM}/day) during monitoring. The participants had the mean age 26.6±7.2 years (range 20–46 years), weight 74.6±12.6 kg, height 1.78±0.09 m, Body Mass Index (BMI) 23.4±3.0 kg/m², body fat 19.2±6.4 % (measured in 11, estimated in 11), fat-free mass 60.2±10.5 kg, VO_2peak 49.3±7.4 mL/kg/min (range 33–65 mL/kg/min). Two participants had repeated monitoring sessions on separate weeks; these were linked to a single person identifier so that their sessions shared one random intercept.

2.2. EA computation

Daily EA was computed as EA = (EI − EEE) / FFM. Daily Energy Intake (EI) was obtained from smartphone food diaries. Days with reported EI below 30 % of the participant's mean were excluded as they are likely under-reported. Fat-Free Mass (FFM) was derived from measured body-fat percentage when available, otherwise estimated from the Deurenberg equation on BMI, age and sex [10].

Exercise energy expenditure for endurance activities (running, cycling and related) was estimated from a CPET-calibrated intensity-zone model. Each participant's ventilatory thresholds VT1 and VT2 and peak oxygen uptake VO_2peak defined three zones with representative VO_2 of 0.9 × VT1, the mean of VT1 and VT2, and the mean of VT2 and VO_2peak; zone-specific caloric equivalents came from respiratory exchange ratio–based conversion factors [11]. Non-endurance activities used MET values from the Compendium of Physical Activities [12].

2.3. Nocturnal HRV processing

Single-lead ECG was processed inside participant-reported sleep windows. RR intervals outside the physiological range 300–2,000 ms were rejected, and a local median filter (±5 surrounding beats, 20% deviation threshold) removed remaining ectopic beats and artefacts. The cleaned series was segmented into 5-minute windows requiring at least 4 minutes of valid data. Time-domain parameters (MeanRR, SDNN, RMSSD, lnRMSSD, pNN50, SDHR) and frequency-domain parameters (LF power 0.04–0.15 Hz, HF power 0.15–0.40 Hz, LF/HF ratio) were computed per window and averaged across the night, using Welch's periodogram on RR series resampled to 4 Hz for the frequency domain [3]. The night immediately following EA day was paired with this day EA value.

2.4. Statistical analysis

For each HRV parameter, a linear mixed-effects model (LMM) was fitted of the form

$$HRV_{ij} = \beta_0 + \beta_1 \cdot EA\_binary_{ij} + u_j + \varepsilon_{ij},$$

where HRV_{ij} denotes the value obtained on night i of participant j, EA_binary_{ij} is the energy availability category of the preceding day (0 = LEA, 1 = adequate EA), β_0 is the fixed intercept corresponding to LEA nights, β_1 is the fixed effect of adequate EA, $u_j \sim N(0, \sigma^2_u)$ is the participant-specific random intercept accounting for between-subject differences in baseline autonomic tone, and $\varepsilon_{ij} \sim N(0, \sigma^2_e)$ is the residual error. The random intercept accounts for inter-individual differences in baseline HRV, such that β_1 represents the within-person difference in HRV between Adequate and Low EA days.
Statistical significance was assessed at $\alpha = 0.05$. Standardized within-person effect sizes were computed as $d = \beta_1 / \sigma_e$, where σ_e is the residual (within-participant) standard deviation of the model, i.e. the typical fluctuation of nocturnal HRV between nights within the same participant. The effect size therefore expresses the EA-related difference in units of that fluctuation: d = 1 would mean a difference as large as the night-to-night variability a participant shows anyway, and d = 0.5 a difference half that size. Values around 0.2, 0.5 and 0.8 were interpreted as small, medium and large effects.
For descriptive statistics, HRV values were averaged within each participant and EA category and then across participants, so that each participant contributes equally regardless of the number of days.

3. Results

3.1. Participants and EA distribution

The 22 participants contributed 123 person-day observations: 71 Low days (mean EA 19.6±9.0 kcal/kg^{ffM}/day, range −9.5−29.5) and 52 Adequate days (mean EA 51.1±5.8, range 45.0−69.8). Each participant contributed on average 3.2 Low and 2.4 Reported energy intake was 2,485±761 kcal/day on LEA days and 3,111±694 kcal/day on Adequate EA days.

3.2. Within-subject effect of EA on

nocturnal HRV

Table 1 summarizes the descriptive HRV values and the p-values for the Adequate-versus-Low EA contrast; Table 2 gives the corresponding model coefficients, 95% confidence intervals (CI) and standardized effect sizes. Eight of nine HRV parameters differed significantly at α=0.05. Individual changes for four selected parameters are shown in Figure 1. Adequate EA days were associated with higher parasympathetic and overall variability indices, most clearly RMSSD (+9.4 ms, p=0.005), pNN50 (+6.3 percentage points, p=0.003) and HF power (+258 ms², p=0.004); LF power, SDNN, SDHR, meanRR and lnRMSSD followed the same direction (all p≤0.017).

Table 1. Descriptive nocturnal HRV values on Low and Adequate EA days and p-values for the within-subject contrast (123 person-days from 22 participants; Low n = 71, Adequate n = 52). Values are means±SD of the participant means; p-values are from LMMs with random intercepts on unique individuals. Coefficients and effect sizes are in Table 2. *p < 0.05, **p < 0.01

Parameter	Low EA mean±SD	Adequate EA mean±SD	p
lnRMSSD	3.98±0.39	4.11±0.34	0.014*
SDNN (ms)	88.3±17.6	96.0±15.9	0.011*
RMSSD (ms)	60.8±22.8	68.4±22.4	0.005**
pNN50 (%)	32.4±15.9	38.2±15.4	0.003**
MeanRR (ms)	1,104±165	1,134±169	0.017*
SDHR (bpm)	5.05±1.07	5.39±1.14	0.007**
LF (ms²)	1,555±537	1,805±535	0.012*
HF (ms²)	780±625	957±689	0.004**
LF/HF	3.60±2.71	3.37±2.94	0.303

Table 2. Within-subject LMM estimates (Adequate-Low EA) in the original units with 95% CI (123 person-days from 22 participants). Standardized effect sizes d are defined in Section 2.4. p-values are in Table 1.

Parameter	Adequate-Low EA (95% CI)	d
lnRMSSD	+0.133 (0.027, 0.239)	0.52
SDNN (ms)	+7.91 (1.81, 14.00)	0.53
RMSSD (ms)	+9.40 (2.89, 15.92)	0.60
pNN50 (%)	+6.34 (2.11, 10.57)	0.62
MeanRR (ms)	+29.7 (5.3, 54.1)	0.51
SDHR (bpm)	+0.44 (0.12, 0.75)	0.57
LF (ms²)	+290 (64, 516)	0.52
HF (ms²)	+258 (81, 435)	0.60
LF/HF	−0.20 (−0.57, 0.18)	−0.22

Tonic heart-rate metrics showed weaker responses: meanRR was longer on Adequate EA days (+29.7 ms, p=0.017). The LF/HF ratio did not differ significantly (p=0.303), which is consistent with proportional increases in both LF and HF power.

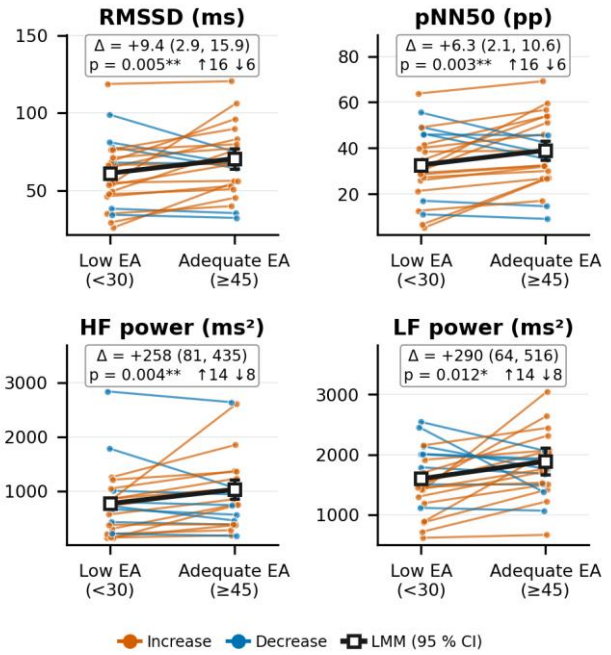

Figure 1. Within-subject change in four nocturnal HRV parameters (RMSSD, pNN50, HF power and LF power) between Low and Adequate EA days (123 person-days from 22 participants). Each thin line joins one participant's mean on their Low EA days to their mean on Adequate EA days; orange marks an increase, blue a decrease, and the arrows give the number of participants in each direction. The bold black line is the fitted LMM with its 95% CI; the box in each panel gives the model estimate Δ with its 95% CI and the p-value. *p < 0.05, **p < 0.01.

4. Discussion

This within-subject analysis shows that day-to-day variation in EA tracks a coherent and quantitatively substantial shift in next-night nocturnal HRV in free-living physically active individuals. Eight of nine HRV parameters were significantly lower after LEA days than after the same individual's Adequate EA days, and the differences are not marginal: adequate EA nights carried on average 15 % higher RMSSD, 19 % higher pNN50 and 33 % higher HF power than that person's own LEA nights. These correspond to medium-sized within-person effects (d = 0.51–0.62; Table 2), which is notable for a single day's dietary exposure observed under free-living conditions. Because the design compares each individual against themselves on different days, inter-individual differences in baseline autonomic tone, fitness, body composition, age and sex are absorbed by the random intercept and cannot

drive the observed effects.

The pattern of responses is physiologically coherent. The three parasympathetic markers (RMSSD, pNN50, HF power) showed the largest increases from Low to Adequate EA days, consistent with enhanced vagal modulation during adequate energy supply, while SDNN and LF power increased in parallel without any change in the LF/HF ratio – both branches upregulated rather than a shift in sympathovagal balance. This would fit the proposed mechanism in which LEA activates the hypothalamic–pituitary–adrenal axis and suppresses autonomic outflow [8,13], and the effect is detectable during sleep, normally a state of maximal parasympathetic dominance [5,6].

The practical reading of these effect sizes is that inadequate EA exacts a systematic autonomic cost on the following night. A d of 0.6 places the EA-related shift at roughly six tenths of the fluctuation an individual shows from night to night anyway, so Low and Adequate nights still overlap within a participant. Repeated nocturnal recordings can nevertheless reveal the difference, in the regime in which wearable HRV monitoring is already used. Thus, the response is already detectable on a day-to-day timescale, not only after months of chronic deficiency. Because LEA days are typically also training days (mean exercise energy expenditure 1,237 vs 159 kcal on Adequate EA days), the present contrast cannot fully separate EA from the acute autonomic cost of the preceding exercise bout; both act in the same direction and both are components of the recovery state that nocturnal HRV is used to track. Further limitations are the observational design, self-reported food diaries, the small cohort and sleep timing without actigraphy. Replication in larger cohorts with controlled EA perturbations and endocrine profiling is needed.

5. Conclusion

In a free-living cohort of physically active individuals, a single day of LEA was followed by a measurably blunted night of cardiac autonomic activity in the same person: vagally mediated variability (RMSSD, pNN50, HF power), overall variability (SDNN, LF power), SDHR and MeanRR were all lower than after that individual's own Adequate EA days, with medium effect sizes on the within-person scale. Covering the energy cost of a given day therefore appears to be a relevant condition for the parasympathetic recovery that follows it, and it operates on a next-night timescale rather than only over months of chronic deficiency. Nocturnal HRV cannot substitute for direct assessment of EA in an individual, but it carries a reproducible signal about recent energy status, and repeated nocturnal recordings are a plausible route to tracking it.

Acknowledgments

This work was supported by Horizon KDT JU project DistriMuSe (grant agreement No. 101139769-2) and co-funded by the Ministry of Education, Youth and Sports of the Czech Republic (grant No. 9A24005).

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